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Sequence of retrograde atrial activation of the human heart[1]
Correlation with P wave polarity

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Studies were performed in 6 patients during open heart surgery to correlate the relative sequence of atrial activation with the polarity of the retrograde P wave in electrocardiographic leads II, III, and aVF. Bipolar atrial electrograms were recorded from selected sites during threshold pacing from sites low on the right side of the atrial septum which when paced resulted in the inscription of either negative or positive P waves in electrocardiograph leads II, III, and aVF. When the atria were paced from a site just anterior to the coronary sinus ostium (negative P waves), the posteroinferior left atrium was activated early and Bachmann's bundle late during the inscription of the P wave. When the atria were paced from a site 2 cm anterior to the coronary sinus ostial site (positive P waves), Bachmann's bundle was activated early during the P wave and the posteroinferior left atrium was activated relatively later. These data from man are consistent with previous studies in the canine heart. Therefore, we deduced that in man during the inscription of negative retrograde P waves in leads II, III, and aVF, atrial activation occurs primarily in a retrograde fashion. However, during the inscription of positive retrograde P waves in man, activation occurs rapidly up the interatrial septum (we believe via the anterior internodal pathway) to Bachmann's bundle, from where it then spreads in a manner similar to that which occurs during normal sinus rhythm.

It previously has been shown both in the experimental animal (Scherf and Shookhoff, 1925; Borman and Meek, 1931; Brumlík, 1958; Moore *et al.*, 1967, 1971; Waldo *et al.*, 1971, 1975) and in man (Daniélopolu and Proca, 1926; Gallavardin and Veil, 1928; McGuire and Rosenberger, 1931; Dressler and Roesler, 1956; Latour and Puech, 1957; Gonzalez Videla, 1968; Waldo *et al.*, 1968, 1970) that retrograde activation of the atria may result in the inscription of positive and biphasic (−, +) as well as negative P waves in electrocardiographic leads II, III, and aVF. Detailed studies of the sequence of activation in canine atria (Waldo *et al.*, 1975) showed that when the retrograde P wave was negative, the atria were activated primarily in a retrograde fashion. However, when the retrograde P wave was positive in those same leads, activation of the atria was such that the wave of excitation travelled rapidly up the interatrial septum to Bachmann's bundle from which point it spread inferiorly over a significant portion of both atria in a manner similar to that which occurred during sinus rhythm. The time of arrival of the impulse at Bachmann's bundle relative to its arrival at the posteroinferior left atrium was critical in determining the polarity and morphology of the P wave. Positive retrograde P waves were associated with early activation of Bachmann's bundle and later activation of the posteroinferior left atrium. Negative retrograde P waves were associated with relatively early activation of the posteroinferior left atrium and later activation of Bachmann's bundle. Thus, the relative activation times at Bachmann's bundle and the posteroinferior left atrium provide markers for understanding the sequence of atrial activation. The present study was conducted to determine if these experimental observations in the dog hold true for man as well.

It has been shown earlier that when the human atria from the region of the coronary sinus ostium

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are paced, negative P waves or negative P waves with a small late positive component in electrocardiographic leads II, III, and aVF are produced (Waldo *et al.*, 1970). When the atria are paced from sites in the AV junction increasingly more anterior relative to the coronary sinus ostium, biphasic (−, +) and finally frankly positive P waves in leads II, III, and aVF are produced (Waldo *et al.*, 1970). In the present study in patients, the atria were paced from two AV junctional sites, one from which pacing produced negative retrograde P waves and the other from which pacing produced positive retrograde P waves in leads II, III, and aVF. The relative sequence of atrial activation during inscription of these P waves was then studied and compared with previous observations in the canine heart.

Methods

Using previously reported intraoperative cardiac electrophysiological techniques (Waldo *et al.*, 1968, 1970, 1973), 6 patients between 8 and 56 years of age were studied during open heart surgery. Five patients had secundum atrial septal defects and one patient had a primum atrial septal defect. No patient had any conduction abnormality in the electrocardiogram before or after the open heart surgery. Observations were made in each case at normothermia during cardiopulmonary bypass after the repair of the atrial septal defect. For each patient, bipolar electrograms were recorded from Bachmann's bundle at a point overlying the interatrial septum, from the sulcus terminalis at a point just below the sinus node, from just within the coronary sinus ostium, and from posteroinferior left atrial sites (Fig. 1 and 2). Electrograms from the sulcus terminalis site (ST) were recorded in all 6 patients from a plaque electrode (Waldo *et al.*, 1968, 1970) sewn there before cardiopulmonary bypass. Electrograms from the Bachmann's bundle site (BB) were recorded from a plaque electrode sewn there before cardiopulmonary bypass in 4 patients, and from a catheter electrode sutured securely before cardiopulmonary bypass to hold the electrode at this site in 2 patients. Electrograms were recorded from the coronary sinus ostium and the posteroinferior left atrial sites in 5 patients by inserting a multipolar catheter electrode into the coronary sinus ostium; the most proximal electrode pair (CSp) was located just within the coronary sinus ostium, a second electrode pair (CSm) was 1·0 cm distal to the proximal pair, and a third electrode pair (CSd) was 1·8 cm distal to the middle electrode pair. The interelectrode distance for each pair of electrodes was 2 mm. This multipolar

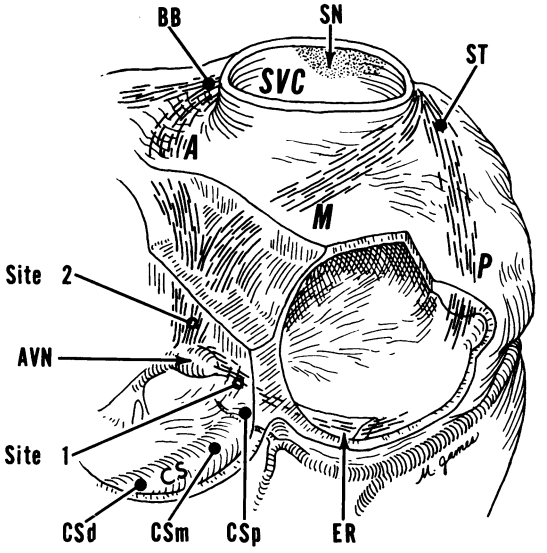

Fig. 1 *This drawing is a partial view of the heart from above and behind the left atrium with parts of the right atrium and interatrial septum cut away. The black dots in the coronary sinus (CS) indicate the distal (CSd), middle (CSm), and proximal (CSp) coronary sinus catheter electrode recording sites. The CSd and CSm sites record activation from the posteroinferior left atrium. Other black dots indicate Bachmann's bundle (BB) and sulcus terminalis (ST) electrode recording sites. The anterior (A), middle (M), and posterior (P) internodal pathways and the interatrial pathway which courses through Bachmann's bundle are schematically depicted sweeps of broken lines. The relation of pacing sites (open circles) 1 and 2 to the internodal pathways is clearly shown. Additional abbreviations: AVN=atrioventricular node; SN=sinus node; SVC=superior vena cava; ER=Eustachian ridge.*

electrode catheter was placed in the coronary sinus during cardiopulmonary bypass via the atriotomy in the free wall of the right atrium created for the repair of the atrial septal defect. It has been shown earlier that this type of atriotomy does not affect P wave polarity or morphology when pacing from low atrial septal sites (Waldo *et al.*, 1970). In one patient, the multipolar catheter electrode was placed epicardially to record from the posteroinferior left atrial sites by positioning the catheter just superior to the course of the coronary sinus and directly below the inferior pulmonary veins. The catheter was quite stable in this position as it was sandwiched securely between the pericardium and the heart. In this position the most proximal electrode pair was located on the epicardium just posterior to the inferior vena cava, such that the CSp site using this

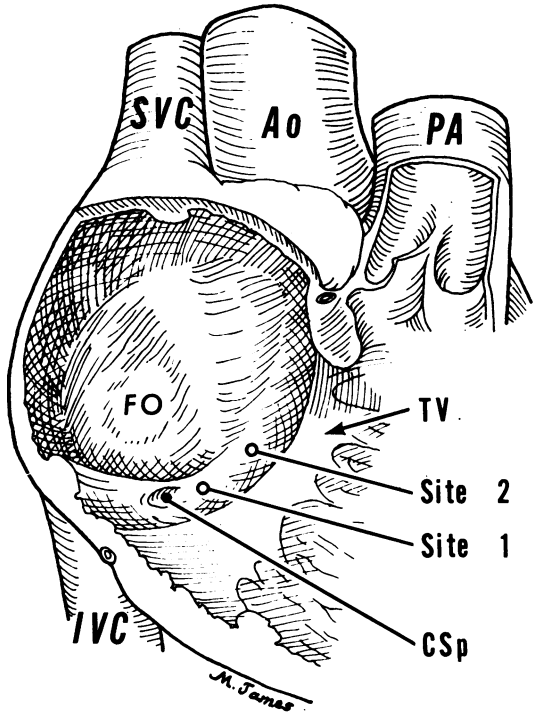

Fig. 2 *In this drawing, the free walls of the right atrium and right ventricle have been removed. Pacing site 1 is just anterior to the coronary sinus ostium and pacing site 2 is 2 cm anterior to pacing site 1. The proximal coronary sinus electrode site (CSp) is represented by the black dot which lies just within the coronary sinus ostium. Additional abbreviations: Ao=aorta; PA=pulmonary artery; SVC=superior vena cava; IVC=inferior vena cava; TV=tricuspid valve; FO=fossa ovalis.*

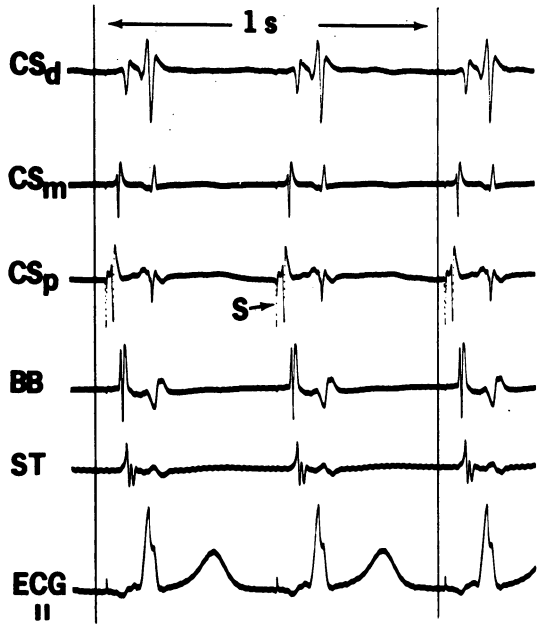

Fig. 3 *Electrocardiograph lead II recorded from one patient simultaneously with electrograms from the five standard recording sites during a rhythm in which the atria are paced from site 1. S=stimulus artefact. See text for discussion.*

technique approximated the CSm site using the technique in which the catheter electrode was inserted into the coronary sinus. Similarly, with the epicardial placement of the catheter electrode, the CSm and CSd epicardial sites were correspondingly more distal relative to the coronary sinus ostium than those sites for the catheter electrode placed within the coronary sinus.

For each study the atria were paced with bipolar threshold stimuli delivered through a hand-held electrode probe from two sites low in the right atrial septum (Fig. 1 and 2). One site (No. 1) just anterior to the coronary sinus ostium was selected because pacing from this site produced P waves which were either entirely negative or negative with a very small late positive component in leads II, III, and aVF (Waldo *et al.*, 1970). The other site (No. 2), about 2·0 cm anterior to the coronary sinus ostium, was selected because pacing from this site produced positive P waves in leads II, III, and aVF (Waldo *et al.*, 1970). The rate at which the atria were paced was just fast enough to achieve stable atrial capture, and generally was 10 beats/min faster than the spontaneous rate.

Results

The results were consistent in all patients. Table 1 contains the conduction times to selected recording sites during atrial pacing. Case 6 had an ostium primum atrial septal defect and thus we could pace only from site 1 (Waldo *et al.*, 1973). Data from two representative studies in which a plaque electrode was sewn to the Bachmann's bundle site and in which the multipolar electrode was inserted into the coronary sinus will be shown.

Fig. 3 illustrates the records from case 5 when site 1 just anterior to the coronary sinus ostium was paced. The P wave in lead II was primarily negative with a small late positive component (Waldo *et al.*, 1970). Conduction time to the proximal coronary sinus electrode site was 21 ms,

Table *Conduction times (ms) to selected recording sites*

Case No.	*Pacing site*	*BB*	*CSp*	*CSm*	*CSd*
1	1	87	29	40	56
	2	45	104	120	104
2	1	58	—	51	72
	2	43	62	81	90
3	1	63	—	16	33
	2	30	34	50	73
4	1	87	13	39	62
	2	68	73	99	123
5	1	45	21	37	60
	2	25	29	43	69
6	1	48	—	14	25
	2	—	—	—	—

to the middle coronary sinus electrode site 37 ms, to the Bachmann's bundle site 45 ms, and to the distal coronary sinus and sulcus terminalis sites 60 ms each. When in the same patient the atria were paced from the low atrial septal site 2 cm anterior to the coronary sinus ostium (site 2), a positive P wave was inscribed in leads II, III, and aVF (Fig. 4). Conduction time to the Bachmann's bundle site then was 25 ms, i.e. 20 ms shorter than when the other site was paced. Conduction time to the proximal coronary sinus electrode site was 29 ms, i.e. this site was activated after the Bachmann's bundle site. Furthermore, both sites were activated before the onset of the inscription of the P wave in leads II, III, and aVF. Conduction time to the middle coronary sinus electrode site was 43 ms or 18 ms longer than to the Bachmann's bundle site. Conduction time to the sulcus terminalis site was 43 ms.

Fig. 5 and 6 illustrate records from case 3. Pacing from site 1 just anterior to the coronary sinus ostium again resulted in negative P waves in leads II, III, and aVF. The electrode probe used to pace the atria was actually in contact with the proximal coronary sinus electrode, which explains the movement artefact in the electrogram recorded from the proximal coronary sinus site. Parenthetically, this occurred also in cases 2 and 6 (Table). Conduction time to the middle coronary sinus electrode site was 16 ms and to the distal coronary sinus site 33 ms. By using the left time line as reference, it is clear that the conduction time to the Bachmann's bundle site (63 ms) was much longer than that to the

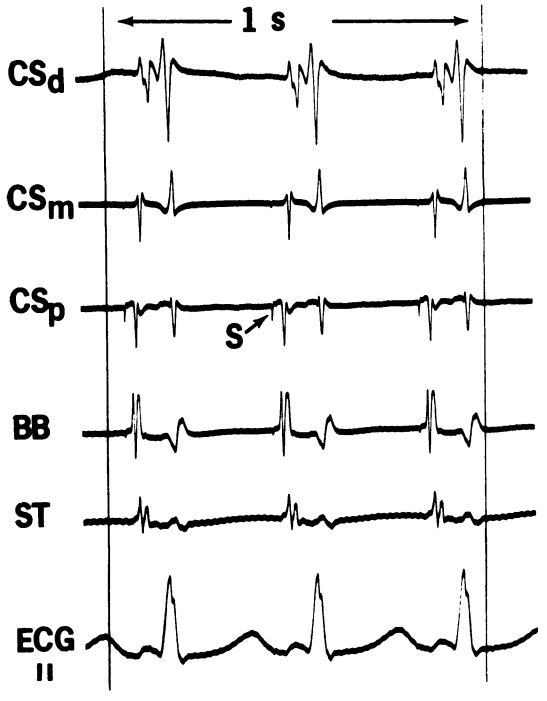

Fig. 4 *Electrocardiogram lead II recorded simultaneously with the atrial electrogram from the five standard recording sites during a rhythm in which the atria are paced from site 2 (same patient as in Fig. 3). See text for discussion.*

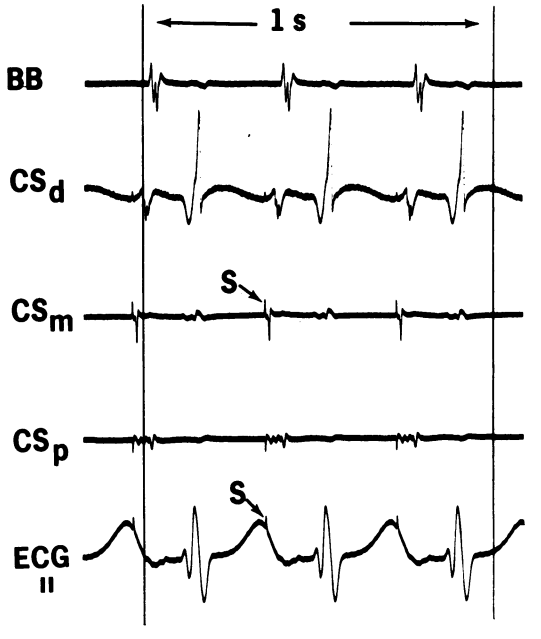

Fig. 5 *Electrocardiogram lead II recorded from a second patient simultaneously with four atrial electrograms (Bachmann's bundle and the three coronary sinus sites) during a rhythm in which the atria are paced from site 1. See text for discussion.*

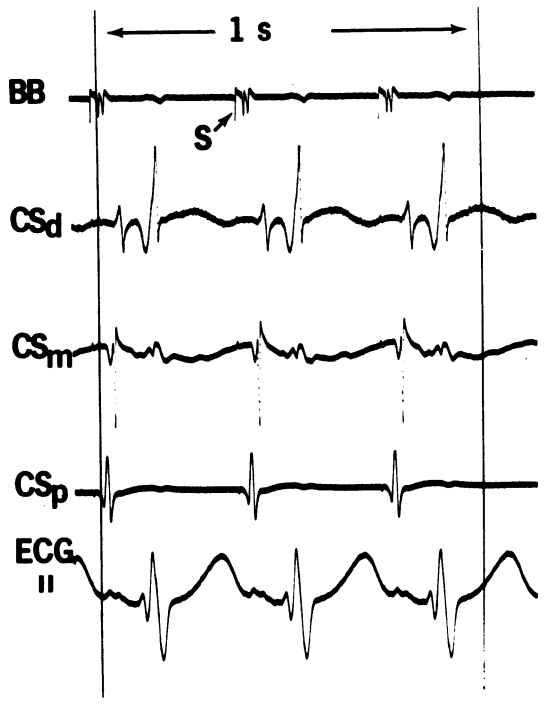

Fig. 6 *Electrocardiogram lead II recorded simultaneously with the same atrial electrograms recorded from the four sites as in Fig. 5 (same patient) during a rhythm in which the atria are paced from site 2. See text for discussion.*

coronary sinus sites. Also, activation of the Bachmann's bundle site was quite late relative to the inscription of the P wave. When in the same patient the atria were paced from the low atrial septal site 2 cm anterior to the coronary sinus ostium (site 2), the P waves in leads II, III, and aVF were positive. Conduction time to the Bachmann's bundle site was then only 30 ms, and again, by using the time line on the left as reference, it can be seen that this site was activated distinctly before inscription of the P wave (Fig. 6). Conduction time to the most proximal site in the coronary sinus was 34 ms or longer than to the Bachmann's bundle site. Conduction time to the middle coronary sinus site was 50 ms and to the distal coronary sinus electrode site 73 ms.

In cases 1 and 4, the stimulus-to-onset of the inscription of the P wave (SP) interval when pacing from site 2 was 40 ms and 48 ms, respectively. When pacing the atria, an SP interval is normally present, though this interval in these two patients is in the upper range of the previously observed stimulus-to-P intervals (Waldo *et al.*, 1970; MacLean *et al.*, 1975). We believe that this in part explains the relatively long conduction times to the selected recording sites (Table). In addition, an interesting finding in case 1 was that the CSd site was activated before the CSm site and virtually simultaneously with the CSp site when pacing from site 2 (Table). Case 1 was the only patient in whom the CS sites were recorded with the epicardially placed catheter electrode. We believe that the distal electrode pair (CSd) probably was located somewhat superiorly to the CSd site recorded from in the other patients and with the inscription of a positive retrograde P wave, this site very likely was activated by a wave-front coming down from above (as we would predict) rather than from a retrograde wave-front coming up from below. When pacing from site 1 in case 1, during the inscription of negative retrograde P waves, the CSd site was activated later than both the CSp and CSm sites, very likely by a retrograde wave-front (as we would predict). Thus, the conduction times to the selected atrial recording sites in this patient as well as the other 5 patients are well explained by our hypothesis regarding the sequence of atrial activation during the inscription of either positive or negative retrograde P waves.

Discussion

COMPARISON OF RETROGRADE ACTIVATION IN HUMAN AND CANINE HEART

The relative sequence of atrial activation during atrial pacing from low atrial septal sites which results in either negative or positive P waves in leads II, III, and aVF in man is similar to that shown in previous experimental studies in the dog (Moore *et al.*, 1971; Waldo *et al.*, 1971, 1975). Since the relative sequence of atrial activation during the inscription of retrograde P waves has been correlated with the actual sequence of activation of both the entire right and left atrium for the canine atria and since the relative sequence of atrial activation during the inscription of these P waves is the same for man as for the canine heart, it is logical to make certain assumptions about the actual sequence of atrial activation in the human heart. When negative retrograde P waves are produced in leads II, III, and aVF, the early arrival of the impulse low in the left atrium and its later arrival at Bachmann's bundle reflects a retrograde sequence of activation within the two atria. Similarly, when positive retrograde P waves are produced in II, III, and aVF, then the arrival of the impulse at Bachmann's bundle earlier than at low left atrial sites reflects the rapid retrograde spread of the impulse up the interatrial septum to Bachmann's bundle,

which we believe occurs via the anterior internodal pathway (Fig. 1). From Bachmann's bundle the impulse then spreads to depolarise significant portions of both atria in a manner similar to that which occurs during spontaneous sinus rhythm.

This interpretation of atrial activation in man not only fits the present data but also fits with previous studies of retrograde P wave polarity in man. If the retrograde positive P waves depends, as we have stated, on rapid retrograde spread of the impulse up the interatrial septum to Bachmann's bundle, then the lesion in patients with ostium primum atrial septal defects should preclude this from occurring and no positive retrograde P wave should be possible in these patients. This, in fact, was shown by a previous study (Waldo *et al.*, 1975) as well as the present study in which low atrial septal sites were paced in patients with ostium primum atrial septal defects and only negative retrograde P waves in leads II, III, and aVF were produced. In contrast, as shown by a previous study (Waldo *et al.*, 1970) as well as the present study in patients with ostium secundum or sinus venosus atrial septal defects or with an intact atrial septum, i.e. patients in whom the main route of conduction in the interatrial septum is intact, positive as well as negative retrograde P waves were produced when low atrial septal sites were paced.

'RETROGRADE' POSITIVE P WAVES

The term 'retrograde' positive P waves may seem a semantic contradiction by usual electrocardiographic concepts. However, it is clear that positive P waves in leads II, III, and aVF can occur in man during activation of the atria from low atrial sites, and during such pacing at least the initial spread of atrial activation necessarily is retrograde. Therefore, it seems reasonable to use this term, particularly as it emphasises the difference between it and retrograde negative P waves.

Since there are so few reported cases of positive retrograde P waves during spontaneous ectopic rhythms (Daniélopolu and Proca, 1926; Gallavardin and Veil, 1928; McGuire and Rosenberger, 1931; Dressler and Roesler, 1956; Latour and Puech, 1957; Gonzalez Videla, 1968), one may ask whether such P waves are anything more than a rare curiosity. It has been long and widely believed that retrograde activation of the atria necessarily results in negative P waves in leads II, III, and aVF (Lewis, 1921), but our results suggest that one also must consider the possibility that an ectopic rhythm with positive P waves in II, III, and aVF could represent 'retrograde' activation of the atria. There is evidence to suggest that such spontaneous rhythms are possible. For example, the low atrial septal sites which when paced result in retrograde positive P waves in II, III, and aVF are in close proximity to the internodal pathways (James, 1963; Merideth and Titus, 1968). Since specialised cells in these pathways have been shown to be capable of spontaneous impulse formation (Hogan and Davis, 1968), then ectopic rhythms which may be initiated from such a site could be expected to produce positive retrograde P waves. The rhythm formerly known as coronary nodal rhythm may be one example, since it is associated with positive P waves in leads II, III, and aVF and with a short PR interval (Langendorf *et al.*, 1944; Brumlík, 1958; Waldo *et al.*, 1970). In a classic review of paroxysmal atrial tachycardia (Barker *et al.*, 1943), fully one-third of these rhythms were said to have positive P waves in II, III, and aVF. Since that time, it has become generally accepted that paroxysmal atrial tachycardia is an AV nodal re-entrant rhythm (Goldreyer and Bigger, 1971). Depending upon the emergence and propagation of atrial septal activation, some of these re-entrant rhythms could be expected to have positive P waves in II, III, and aVF.

Although it is very likely true that there are spontaneously occurring sinus node re-entrant rhythms, possibly including some of the rhythms described above (Barker *et al.*, 1943), all the suggested criteria (Narula, 1974) for the diagnosis of this rhythm (positive P waves in leads II, III, and aVF similar to the P waves which occur during sinus rhythm, and a relative sequence of atrial activation such that a site high in the right atrium is activated before a site low in the right atrium) were fulfilled in the present study when we paced the atria from the low atrial septum (site 2). While our observations do not disprove the concept of sinus node re-entrant rhythms, nor is it our intent to suggest that sinus node re-entrant rhythms do not occur, it is clear that the criteria so far proposed for the diagnosis of such rhythms also are fulfilled by rhythms originating low in the atrial septum (our site 2). To know with certainty the site of origin for re-entrant supraventricular arrhythmias having positive P waves in leads II, III, and aVF, additional criteria will have to be established.

SPECIALISED ATRIAL CONDUCTION

During the inscription of retrograde positive P waves in II, III, and aVF, the spread of activation up the interatrial septum in these human atria was so rapid that the Bachmann's bundle site was depolarised before the proximal coronary sinus site (CSp), i.e. the site just within the coronary sinus ostium and also usually before the inscription of the P wave. Since the distance from site 2 to the

Bachmann's bundle site is more than twice as long as the distance from site 2 to the CSp site (Fig. 1), we believe these data further support the accumulated evidence summarised recently (MacLean *et al.*, 1974) favouring functionally important specialised atrial pathways in both human and canine hearts.

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